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PATHOLOGY AND TREATMENT OF PARALYSIS FROM POTT'S DISEASE.

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PATHOLOGY AND TREATMENT OF PARALYSIS FROM POTT'S DISEASE.[1]

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AT first sight one is likely to regard the paralysis that so frequently occurs in spinal caries as the result of direct pressure upon the cord by the bent and distorted spinal column. Reflection as to the history and symptoms of the disease shows that this is not generally the case, and this is also proved by the conditions found on the post-mortem table. The onset of paralysis in Pott's disease is almost always gradual, and it is a well-known fact that cases are not unusual in which an acute angular curvature has existed for many years without any symptoms of paralysis, and yet, eventually, paraplegia may develop.

In a patient who was under my care there had been a prominent antero-posterior curvature in the dorsal region for many years without interfering with the man's locomotion. One day while carrying a heavy tin box and some fishing-tackle he ran across a ploughed field to catch a train. When he reached the train he felt considerable

[1] Read before the Section on Orthopedic Surgery of the College of Physicians, October 20, 1893.

numbness in his legs, and upon arriving home he walked up stairs with difficulty. In twenty-four hours he was unable to stand, and within a few days there was complete paraplegia that lasted several weeks. The patient ultimately entirely recovered after suspension and the application of a plaster-of-Paris jacket.

Were the paralysis the result of bony pressure there would be complete loss of motion and sensation below the point of injury, just as is met with after fracture of the vertebræ or fracture-dislocation; but it is well known that the paralysis in Pott's disease is generally limited to motion.

The relatively large caliber of the spinal canal, compared with the size of the cord, permits of a considerable amount of bending of the spinal column without materially impinging upon the cord. Of course, it does sometimes occur that in an angular curvature there is also displacement of one of the bodies of the vertebræ, followed by complete compression of the cord, but this is not often the case. The spinal cord shows extraordinary tolerance of pressure, if the pressure is gradually produced. It is not an unusual thing to find in an old case of Pott's disease that the cord has been flattened almost into a ribbon, without its functions having been impaired to a marked degree.

The papers that are to be read dealing with the surgical pathology of spinal caries render it unnecessary for me to speak in detail of the changes that occur in the bones.

The inflammation of the bodies of the vertebræ and of the inter-vertebral substance is the first step

in the process. This inflammation, which is either tuberculous or scrofulous, results in the breaking down of the substance of the bone, and when this occurs the deformity of the spine follows. The effect of this inflammation is often to cause suppuration, and the pus will either find its way externally or it may penetrate into the spinal canal and there make pressure upon the cord. Frequently, however, no pus is formed. The bodies of the vertebræ soften and break down, undergoing a caseous degeneration, and absorption of the inter-vertebral discs takes place. While these changes are going on there is an extension of the inflammatory process to the dura. The connective tissue between the dura and the bone becomes thickened, with the development of a thick layer of organized lymph between the two, giving rise to what is known as external pachymeningitis. In this semi-purulent form of pachymeningitis, the layer of inflammatory products of caseous or fibrinous material and sometimes with pus in spots, often becomes extraordinarily thick; occasionally it is as much as one-half inch in thickness, and may extend vertically two or three inches.

The inner surface of the dura is usually smooth and unaffected by the inflammation, but there may be an inflammation of the inner side as well, although this is rare. However, when it does occur the internal membranes are also involved, and adhesions take place between them and the dura and the cord.

The pachymeningitis being the result of extension of the inflammation from the contiguous surface of

the degenerating vertebral bodies, the inflammatory thickening takes places on the anterior surface of the cord, the result being pressure upon this region and consequently paralysis of motion. The paralysis of motion is sometimes the result of simple pressure upon the cord, but there may also be a myelitis from an extension of the inflammation, and this may extend all the way through the substance of the cord, giving rise to the so-called "transverse myelitis."

On post-mortem examination the cord presents evidence of the compression to which it has been subjected and, as has been already remarked, may sometimes be flattened out like a strip of tape. It may be reduced in size, so that for an inch or two it is not larger than a lead-pencil. When the cord has been compressed, the color is grayish, and in cases of short duration its consistency is soft, while in cases of long-standing it is firmer than usual. The amount of myelitis does not depend upon the degree of pressure, for a considerable amount of myelitis may be found in cases in which the compression has been slight, extending, as has been already said, from the adjacent inflammation in the dura.

Under the microscope there is found a general increase in the interstitial connective tissue, with various changes in the cells, but late in the disease all of the cells disappear. The nerve-elements also undergo degeneration, the nerve-fibers becoming shrunken and having but a narrow sheath of myelin. When there has been extreme pressure, nothing is to be found but a narrow fibrous band at the point of division, which is sometimes difficult to discover,

but there is never actual division of the cord itself. The nerve-roots also show signs of the effects of the pressure and inflammation where they pass through the membranes and inter-vertebral foramina. They are often compressed by the thickening of the dura, and they may also suffer from the narrowing of the spinal canal. On section they are sometimes red and swollen, or they may be shrivelled and gray in color.

The symptoms in caries of the spine are threefold: first, those of bone-disease; secondly, those of disease of the nerve roots, and thirdly, those of the changes in the cord itself. These latter may be subdivided into the symptoms depending upon myelitis from extension of inflammation and those of compression of the cord. The symptoms referable to the bone-disease properly belong to the surgical aspect of the subject, but they are so important in aiding us to make a correct diagnosis that it is well to bear them in mind. In caries of the spine there are localized pain and tenderness on pressure at a certain point of the spine. This pain is increased by movements of the trunk and more particularly by the bending of the body forward. At the seat of pain there is usually some prominence or irregularity of the spinous processes, although this is not always present, and is a later symptom in the disease.

If the disease is in the cervical region, movements of the head will occasion pain, and, in order to avoid the pain caused by these movements, the patient usually carries the head stiffly or supports the chin with his hand. Should the disease be

situated in the dorsal region there is a compensatory curve in the lumbar region, and any patient presenting a well-marked lordosis, in connection with symptoms of nerve-disturbance in the extremities, should excite our suspicions as to bone-disease, even if it is not possible to detect any deformity in the vertebra. We should not forget that lateral curvature may also be due to caries of the spine and give rise to pressure-symptoms.

Symptoms of disease of the nerve-roots are usually present, although it is stated by some authorities that they are not frequently conspicuous. In my experience, however, there are usually some evidences of irritation or disease of the nerve-roots. These symptoms consist in pain in the distribution of the nerves that emerge at the inter vertebral foramina in the affected region of the cord. There may be pain in the course of the nerves in the limbs, or the symptoms may be only those of paresthesia, such as numbness and formication. There is frequently more or less girdle-pain at the level of the affected nerve-roots, and occasionally herpes zoster may be met with in the course of the irritated nerves. It is not infrequent, in spinal caries, to find pain referred to the epigastrium, and in one or two cases I have seen severe pain in the hypochondriac region, simulating renal colic.

In irritation of the motor roots we have symptoms of disordered muscular action and wasting of the muscles. Occasionally, there are active muscular contractions as the result of the irritation of the motor nerves. When the cervical spine is diseased, there may be disorder of the sympathetic

nerves, with localized sweating and dilatation of the pupils.

The symptoms of the cord-disease, when dependent upon myelitis from extension of the inflammation, are like those of myelitis from any other cause, but, as has been already observed, the myelitis is rarely transverse and is usually confined to the anterior portion of the cord; hence the symptoms are principally motor in character.

The onset of the paralysis is usually gradual and often occurs as the result of some unusual exertion or exposure to cold. The rapidity of development varies and total paraplegia may result in a few days, although it is usually several weeks before it is complete.

The degree of motor paralysis also varies with the extent of the cord-disease; sometimes there is merely stiffening of the limbs, with a spastic gait, with slight impairment in the control of the bladder and rectum, while at other times motor paralysis is absolute. When the paralysis is due to compression, it is likely to be more complete, with sensory disturbances, as well as the loss of motor power. When the pressure is confined to the anterior portion of the cord, as it usually is, there is only motor paralysis, with more or less weakness of the bladder and rectum. The pachymeningitis may, however, entirely surround the cord, and if there is much fibrinous or caseous exudation the compression will be almost total. When there is bony displacement, in addition to the pachymeningitis, it is readily seen that the compression of the cord will be still greater. Under these circumstances, the motor and sensory paralysis

is entire, and there is also paralysis of the bladder and rectum. Bedsores do not generally occur, except as the result of neglect, because, as a rule, the trophic disturbances are not great.

In both forms of cord-disturbance, whether due to myelitis from extension or myelitis from compression, the reflexes are greatly exaggerated, provided the disease is above the lumbar region. The knee-jerks are grossly exaggerated, ankle-clonus is present, and the superficial reflexes are also unduly excitable. Occasionally patella-clonus is present, and the toe-reflex, to which I have called attention elsewhere, may be developed. If the disease is situated as low as the lumbar enlargement the reflexes will be entirely absent and there will be paralysis of the bladder and rectum.

The symptoms, of course, will depend upon the region of the spine in which the disease exists. If it is in the cervical region there may be paralysis of both arms and legs, or if it is in the dorsal region, which is the common seat of caries, there will be simple paraplegia. There is occasionally complete anesthesia, but sometimes this is confined to areas of varying extent over the limbs, and at times we may have associated with loss of tactile sense hyperesthesia. Generally both legs are equally paralyzed, but occasionally one is affected more than the other, and in a patient whose history I shall relate the knee-jerk was exaggerated on one side and abolished on the other side.

Among the complications that may be mentioned are bedsores, cystitis, and contractures. The symptoms may change, owing to the fact that de-

generation of the cord may extend from the original focus of disease. An ascending degeneration of the posterior columns will give rise to tabetic symptoms. Gowers asserts that the inflammation may ascend in the pyramidal tracts when the disease is in the lumbar region, and thus give rise to paralysis of the arms.

The course of the disease, once the paralysis has been established, depends to a great extent upon the condition of the bone. If the bone-disease should heal, the inflammation of the cord usually subsides as the pressure becomes less, although if the myelitis exists independently of pressure this improvement does not necessarily take place. The sensory paralysis when present usually passes away early and the motor loss remains for a longer time. The motor paralysis may remain for months and even years before improvement takes place. In one of the cases that I shall report there was total paraplegia for sixteen months, and yet the patient recovered complete use of the legs. In many cases the paralysis gradually passes away, even when the compression of the cord to a considerable degree remains. Should no improvement take place in the condition of the bones the paralysis is likely to remain complete, and although life may be extended for several years, eventually death occurs as the result of bedsores, cystitis, or from the development of tuberculous disease elsewhere. Relapses may occur during the course of the disease, but they are not common. When improvement has once begun it is usually constant.

The diagnosis of compression-myelitis is easily

made, provided we find symptoms of bone-disease; and in all cases of spastic paraplegia one should carefully examine the spine for irregularities and for tenderness. This is more particularly to be looked for in children, because transverse myelitis or primary lateral sclerosis is unusual at an early age, whereas spastic paraplegia from Pott's disease is comparatively common in children. A mistake frequently made is overlooking the spinal disease in children, and it is a very serious error as regards the prospects of recovery. I have on more than one occasion seen children with paraplegia made to take as much exercise as possible when there was well-marked caries of the spine present which had not been detected, and when every movement caused pain. The symptoms depending on disorder of the nerve-roots also aid us in making a diagnosis.

The diseases most likely to be mistaken for compression-myelitis are chronic myelitis, transverse myelitis, primary sclerosis of the lateral columns, and, when the disease is in the cervical region, progressive muscular atrophy. Another condition for which paralysis from Pott's disease may be mistaken is syringomyelia. In this disease, however, there is no pain, although the spinal column frequently presents a lateral curvature, and there is the well-known symptom of inability to distinguish heat and cold.

We must not forget that there are other causes of pressure than caries and pachymeningitis. There may be tumors of the cord and aneurism.

The prognosis depends upon the extent of the paralysis, and the region of the cord affected; but

there is no disease that gives rise to such complete paralysis of motion and sensation in which we may hope for ultimate recovery, as in paralysis from Pott's disease. Even after the lapse of months or even years, complete paralysis of motion may pass away and the patient become able to walk with his previous activity. Should there be complete palsy of motion and sensation, as well as paralysis of the bladder and rectum, the prognosis is bad, but even in such cases recovery may take place. If the disease is in the lumbar enlargement or in the cervical enlargement, the chances of recovery are less. We must also bear in mind that the prognosis depends upon the prospect of recovery from the constitutional trouble. The scrofulous or tuberculous tendency may be developed elsewhere and the patient die from lung-disease or disease in some other organ.

TREATMENT.—In many cases of paralysis from Pott's disease all that is necessary to restore the power of motion is to give the patient complete rest, and some cases recover even when only a small amount of rest has been taken. The healing of the bone-disease depends very largely upon rest of the part, and the deformity that results is largely dependent upon the position in which the patient has been placed during the healing of the caries. In some cases, even after the inflammation of the vertebræ has apparently entirely ceased, paralysis remains in spite of complete rest having been enforced for many months. It is these cases that come under the care of the neurologist, and it is in these cases, I am happy to say, that the most gratifying results are obtained.

While complete rest is a necessary element in treatment, it must be supplemented by other means. The most important of these is extension of the spine by suspending the body by the head. The most satisfactory way of accomplishing this is to place the patient in an arm-chair, while from the back of the chair is a bent iron rod which projects above the patient's head. To the end of this rod is attached the cord from a head-sling. It is a good plan to insert between the head-piece and the iron bar a spring balance, so that the amount of extension may be measured in pounds. The apparatus I show you is what is usually employed in these cases at the Infirmary for Nervous Diseases.

The patient begins to have extension for fifteen minutes twice a day, and the amount of traction varies from ten to twenty-five pounds, according to the weight of the patient. The degree of pull is gradually increased and may reach seventy pounds. The length of time that the patient is suspended is increased each day until he sits up with the extension on for three hours at a time. The good effects of suspension are occasionally observed immediately, but sometimes no benefit is seen until after several weeks. I have frequently seen cases that have had rest and spinal jackets, and whose paralysis has remained stationary, yet who made immediate gain after the employment of extension.

Extension is sometimes made while the patient is lying in bed by having a cord and weight passing over a pulley at the head of the bed. This is applicable in children, but in adults the weight of the body is too great to make extension in this way effective.

Suspension after the method of Motschutkowski, of Odessa, in the treatment of locomotor ataxia, is not applicable to paraplegia from caries of the spine, as the patient is usually unable to manage the apparatus as well as an ataxic, and besides, by this method the suspension can be maintained for only a few minutes.

Some assert that the good effects from suspension are due to the straightening of the spinal column, but it is doubtful if very much elongation of the spine can be effected by this means. Others declare that by suspension the spinal cord is actually stretched. Reid and Sherrington[1] have made some interesting experiments in connection with the effect of movements of the body upon the size of the spinal canal. They arrive at the following conclusions: First, that when the body hangs freely and vertically from the skull the capacity of the cranio-vertebral canal is at a maximum; secondly, that with the body in this position, when the weight of the trunk and limbs is taken off by lifting and supporting the body vertically there is a diminution in the capacity of the cranio-vertebral canal, but the diminution is a slight one. They also state that it would appear that by suspension the size of the spinal cord is increased in the adult of middle age to the extent of some 100 cubic millimeters.

Althaus thought it probable that in suspension the spinal cord is materially stretched, and by this means some of the adhesions from chronic meningitis are broken down, thus allowing the freer transmission of nerve-influence. In a modified form I am

[1] Brain, 1891.

disposed to accept this view. I can scarcely believe that by suspension the cord can be stretched sufficiently to break down adhesions; but I think there is probably sufficient traction made upon it to influence the adhesions between the dura and the thickened connective tissue, and that thus absorption of the exudation is promoted.

There is also another probable effect of the extension, which is to relieve pressure upon the nerve-roots.

The treatment of Pott's disease by suspension seems to have originated with Professor J. K. Mitchell, in 1826, who wrote a paper on this subject. He used this plan principally in children, and the suspension was effected by placing the child in a frame on rollers, with a curved iron bar extending over the head, from which the patient was partially suspended by the head-sling. This method of treatment seems to have been very successful in the hands of Professor Mitchell, but it fell into disuse, until a few years ago attention was drawn to it by Dr. Benjamin Lee.

In 1889 Dr. S. Weir Mitchell published an account of the treatment of Pott's paralysis by suspension.[1] He reviewed the treatment of his father, Professor J. K. Mitchell, and related his own experience.

I had the privilege of seeing many of the cases that he treated by extension at the Infirmary for Nervous Diseases, and the admirable results that he attained have given me great confidence in this plan of treatment.

Dr. Weir Mitchell's conclusions in regard to suspension are that, by means of it, the spinal deformity is greatly lessened; that it should be used early in Pott's disease, and that no case of Pott's disease ought to be considered desperate without its employment. In regard to the mode of action of suspension he believes that the "pull probably acts more or less directly on the cord itself, and that the gain is not explicable merely by obvious effects upon the mechanical bony curve."

[1] American Journal of the Medical Sciences, May, 1889.

Dr. Leroy W. Hubbard read a paper before the New York Academy of Medicine, March, 1890, on "The Use of Suspension in Pott's Disease," and spoke very favorably of it.

Dr. Putzel, in the discussion on Dr. Hubbard's paper, said that his pathologic studies had led him to believe that the majority of cases of Pott's paralysis were not due to pressure, but to transverse myelitis, and in his experiencewith suspension he considered it a method that was at best of only temporary relief.

Dr. Birdsall thought that the improvement in paraplegia from Pott's disease was due to the slight separation of the vertebræ and consequent improvement in circulation of the affected parts, particularly the nerve-roots.

Dr. L. C. Gray spoke favorably of suspension in the treatment of paraplegia.

Dr. R. H. Sayre said that, carried to excess, suspension failed to give relief in his experience.

Dr. Phelps considered that suspension should not be used when the vertebral disease was still active.

Dr. E. G. Brackett (*Transactions American Orthopedic Association*, 1891) reported several cases of paralysis from Pott's disease, in which suspension had given good results.

Dr. Benjamin Lee, in the discussion that followed Dr. Brackett's paper, said that he wished to claim for himself whatever honor might attach to having been the first to propose and use self-suspension in the treatment of Pott's disease, lateral curvature, spinal pressure, paralysis, and of the conditions known as spinal irritation. He says that the treatment by suspension is very old, that it was used by Ambroise Paré, and that he got the idea from Hippocrates.

Counter-irritation is useful in many cases as an adjuvant to suspension, and the best way of making this is by the actual cautery, that of Paquelin being the most convenient to use. Sometimes when, in spite of suspension and counter-irritation, the progress of the case is slow, benefit seems to follow the

use of internal remedies that have the power of promoting absorption. I have found potassium iodid useful under these circumstances when given in moderate doses.

Of course, in connection with the means already used, it is of the first importance to improve the general health of the patient by the administration of iron and cod-liver oil, and by giving the best and most nutritious food that can be taken. Massage is a most valuable aid, and it should be practised daily by a person well skilled in its use. By this means the nutrition of the muscles is improved, the circulation is stimulated, and a larger amount of food can be assimilated.

The use of the plaster jacket is of value in connection with suspension, and it is best to have the apparatus cut down in front so that it may be removed at pleasure. When the patient is sufficiently convalescent to go about, the half-tanned leather jacket made by Gemrig is helpful in giving support to the spinal column and protecting it from jars and injuries.

When the paralysis remains stationary, notwithstanding the faithful employment of the means already suggested, the question of surgical interference is worthy of careful consideration. Within the past few years the operation of laminectomy has been performed in many cases for paralysis from spinal caries. The results of this operation have been so successful that, with favorable conditions as to the general health of the patient, the operation should be advised whenever the paralysis has resisted the treatment that I have described. The

mortality in these operations is comparatively small, and the beneficial effects have been shown by the brilliant operations of Macewen and others.

In my opinion, however, the operation of laminectomy is not indicated in cases in which the paralysis is purely motor. The removal of the laminæ will not relieve pressure, and it is difficult, if not impossible, to remove the exudation on the anterior surface of the cord without injury to the cord itself; moreover, it is not easy to determine beforehand whether the motor paralysis is due to pressure or to myelitis. When an operation is decided upon, the neurologist can aid the surgeon by indicating the point at which the operation should be performed, and in determining this the schedule for the localization of the functions of the segments of the spinal cord, given by Starr in his book on the *Familiar Forms of Nervous Disease*, will be found useful.

The following cases illustrate the advantage of extension, combined with rest, in the treatment of paralysis from Pott's disease.

Case I.—John T., aged eighteen, a native of Ohio, was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases, February 16, 1888. He is of healthy family with no history of tuberculosis in any of the members. He had always been strong and healthy until September, 1886, when he was violently bumped against a tree by four of his schoolmates. He suffered acute pain in the spine at the time; the pain was also felt in the epigastrium and radiated thence into the legs and shoulders.

About three weeks after the injury a lump was discovered in the dorsal region of the spine. He became progressively weaker, and by January, 1887,

four months after the injury, he was unable to walk or to sit up. On admission he weighed eighty-four pounds. There was angular curvature at the seventh dorsal vertebra. He could sit up with the aid of a leather spinal jacket which he had worn for several months. He had apparently no power to move the legs. The knees and ankles were in a condition of rigidity; the knee-jerk was exaggerated; ankle-clonus, patella clonus, and the toe-reflex were all present. The condition of trepidation of the legs, known as spinal epilepsy, was readily induced; the plantar reflexes were exaggerated, but no cremasteric or abdominal reflex could be elicited. Sensation was impaired but not lost. There was no loss of the functions of the bladder or rectum. There was a small bedsore over the sacrum.

The patient was ordered rest in bed and massage, and the Paquelin cautery was applied on either side of the angular curvature. At the end of a month suspension was made by seating the patient upright in bed and having the suspension-apparatus projected above the head of the bed. Two months later he began to sit up in a chair while suspended, and about this time he began to have slight power of motion of the toes, and soon after this the power to flex the feet and legs followed.

On July 23d, the patient was able to move his legs, drawing up the knees while lying on his back. Sensation was improved but not yet perfect. In October he began to walk about with crutches, and from this time he steadily gained in power and soon dispensed with his crutches, but still continued to wear his spinal jacket.

I had a letter from this patient a short time ago, in which he tells me that he has remained perfectly well and that he has a position of "motor-man" on an electric car in Cleveland, Ohio. He says that he

works fourteen hours a day and is as active as anyone he knows, as he is able to jump off and on the car while it is in motion, and that he can run as well as he ever could.

In this case it will be observed that the paralysis became complete in January, 1887, and it was not until May, 1888. sixteen months later, that power of motion began to return.

Case II.—Robert H., aged twenty-one, a shoemaker by occupation, was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases, December 12, 1892. He had been a patient at Dr. Goodman's clinic since March, 1888, and had been treated with a Kolbe brace for spinal caries. The disease had followed lifting a heavy weight.

On admission the patient complained of extreme stiffness in the legs, with pain in the back extending down the legs. The pain was very severe, and the rigidity of the legs almost prevented locomotion. There was no muscular wasting. The knee-jerk on the right side was exaggerated, but on the left side it could not be elicited. There was no clonus.

The patient was ordered massage, rest in bed, and extension for fifteen minutes daily in the chair, the time to be increased by five minutes daily. On December 22d it was noted that the patient was being suspended for one hour daily and that the legs were more supple. On March 25th the patient was sitting up while wearing a plaster jacket, but still complained of severe pain in the back, and for this the cautery was applied. This immediately relieved the pain in the back, but the man still complained of pain in the anterior part of the thighs. On May 7th he was discharged greatly improved. He was able to walk without trouble, had no rigidity of the limbs, and the pain in the back was only occasionally troublesome. There was now knee-

jerk present on both sides; it was exaggerated on the left side, and on the right side it was slightly in excess of the normal reaction.

Case III.—The patient is Daniel C. B., a mulatto, twenty-seven years of age, and was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases, April 10, 1893. His father, mother, and eight brothers are all living, and there is no history of tuberculous or of Pott's disease in the family. He is married and is the father of two healthy children. He never had rheumatism and denies venereal disease.

Nine years ago a curvature in the spine was noticed. He thinks it came from a strain. No abscess formed and the legs were not affected. He was in bed at this time for three months on account of weakness of the back, but the legs were not paralyzed. Since that time he has been steadily going about and doing his work as a janitor until three months ago, when he was taken down with influenza and was in bed for one week. When he got up he was able to walk on the first day; on the second day he became very weak in his legs, and on the third day he was forced to remain in bed. His legs became gradually helpless and there was marked pain, which was of a dull, aching character, in the back and on the left side. For six weeks before admission there had been spasmodic contractions of the flexors of the legs and thighs, lasting for a few minutes, and then he could extend the legs with his hands.

Condition on examination: The patient is a light mulatto, with pallid mucous membranes. He lies on his side in bed with the thighs flexed on the abdomen and the legs on the thighs. There is a marked kyphosis in the dorsal spine, but no scars of abscesses. His teeth are bad; his tongue is foul; the bowels

are constipated, and he has hemorrhoids. There is no paralysis of the bladder or rectum, and, with the exception of a few uric-acid crystals, the urine is normal. There is complete loss of motion in the lower extremities, with the exception of very slight power to move the toes. The legs are in clonic spasm most of the time. Sensation is preserved, but it is slightly delayed. There are no bed-sores, but there is marked wasting of the muscles of the thighs and legs. The knee-jerks are grossly exaggerated; ankle-clonus, the plantar, cremasteric, and abdominal reflexes are present; the toe-jerk on both sides is present.

The patient was placed at rest in bed and was ordered massage and the cautery to the spine; with head-extension, ten pounds for five minutes daily, and increased by five minutes each day. On May 1st, a plaster jacket was applied, to be worn continuously. This has given considerable relief and the patient is able to lift his left leg off the bed. On May 11th he was able to move the toes of the right foot much better. There were still involuntary contractions of the limbs, but these were not so painful. On May 20th, the patient complained of numbness in both legs, from the hips down, and there was impairment of sensation. The patient gradually improved until June 29th, when he again began to lose power in the legs, and from that time until July 23d the legs became almost as helpless as they were when he was admitted. On this date he was ordered potassium iodid, five grains three times a day, and the plaster jacket was reapplied. On August 8th, he was markedly better and from this time the improvement was steadily progressive until October 5th, when the following note was made:

There is no pain, no numbness, or tingling in the

legs; sensation is normal. The movements of both legs are performed well in all directions, but they are weak. He can stand alone, and walks about the ward supported by the wheel-crutch. The plantar, cremasteric, and abdominal reflexes are normal; the knee-jerks are exaggerated and spastic. There is no clonus. The feet and legs are still cold and sweat freely, and although there is still some muscular wasting it is much less than previously.

This case is notable: first, on account of the prominence of the nerve-root symptoms; secondly, because after improvement had set in there was a relapse; and, thirdly, on account of the apparent benefit from the use of the potassium iodid.